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Unusual Case of Acute Flaccid Quadriparesis in South India

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Authors' contributions

This work was carried out in collaboration between both authors. Author JS conceived the idea, evaluated the case and written the manuscript. Author MM contributed for discussion part. Both authors read and approved final manuscript.

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Case Study

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ABSTRACT

Aim: We report an extremely unusual case of thyrotoxicosis presenting as recurrent acute flaccid quadriparesis in a south Indian male. This case is reported to disseminate knowledge about this rare presentation of thyrotoxicosis among medical professionals.
Presentation of Case: This patient presented with sudden onset of symmetrical weakness of both upper and lower limb since early morning with unexplained hypokalemia. He had diffuse toxic goiter with subtle features of hyperthyroidism associated with flaccid quadriparesis. Laboratory investigation revealed marked hypokalemia and hyperthyroidism. He was clinically diagnosed to have hyperthyroidism presenting as thyrotoxic periodic paralysis. He improved with potassium supplementation, beta blocker & anti-thyroid agent.
Discussion: Thyrotoxic periodic paralysis (TPP) may be the initial presentation of thyrotoxicosis in rare occasion. Proximal muscle weakness of lower limb is often the first symptom noted. Hypokalemia noted in TPP is the consequence of a rapid and massive shift of potassium from the extracellular into the intracellular compartment, mainly into the muscles. TPP is distinguished from other forms of periodic paralysis (especially hypokalemic periodic paralysis) with thyroid function tests. Failure to recognize this rare disorder may result in fatal cardiac arrhythmia which is a potential cause of mortality.

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Conclusion: Clinicians should be aware of this atypical presentation of thyrotoxicosis as it is lethal if not treated. Young people with unexplained hypokalemic paralysis even without apparent evidence of thyroid dysfunction should be subjected to thyroid function test to identify this rare disorder.

Keywords: Thyrotoxic periodic paralysis; hypokalemia; potassium; beta blocker.

1. INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is an extremely rare complication seen in uncontrolled hyperthyroidism [1]. Even though it is commonly seen in Asian population, case report from Indian subcontinent is limited. It has an incidence of 2% among Asians with hyperthyroidism as compared to just 0.1 to 0.2% in non-Asians [2]. It is seen in 13-24% of thyrotoxicosis cases in Asians, numerous cases have been reported from Japan and China. It presents as recurrent acute flaccid paralysis in hyperthyroidism due to unexplained hypokalemia. Most common thyroid disorder associated with this disorder is Graves' disease [3]. Many affected patients do not have obvious symptoms and signs of hyperthyroidism. There are many clinical mimickers of TPP like hypokalemic periodic paralysis, acute inflammatory demyelinating polyneuropathy (Guillain-Barre syndrome). It is often an under-diagnosed condition, though clinical presentation in the form of hypokalemic paralysis without apparent cause for hypokalemia in hyperthyroidism is sufficient to make diagnosis in majority of cases [4].

2. CASE PRESENTATION

A 37 yrs. old male who is a manual labourer, admitted in our hospital with chief complaint of inability to stand and walk since early morning of same day. History of minimal weakness of both upper limbs noted. No History of difficulty in breathing. No history of trauma or fall. No history of insect bite. No history of fever, loose stools, vomiting, abdominal pain or excessive sweating. No history of palpitation, fatigue. Not a known case of diabetes mellitus, systemic hypertension, thyroid disorder, bronchial asthma, No history of drug intake for any illness in the recent past. He is not a smoker or alcohol user. Patient had similar episode with spontaneous recovery one month ago. No history of consanguinity. No history of similar illness in family members.

Patient was conscious, afebrile, with diffuse thyroid swelling. No eye signs of thyrotoxicosis were noted. His pulse rate was 104 per min and regular; blood pressure was 110/ 70 mmHg. Cardio vascular system, Respiratory and abdomen examination were normal. Neurological examination showed normal higher mental functions and cranial nerve examination. Spino-motor system examination showed normal muscle bulk with hypotonia predominantly in both lower limbs. Muscle power in both upper limbs was 4/5 with weak hand grip. Lower limb muscle power showed 3/5 in proximal group, 4/5 in distal group. Deep tendon reflexes were diminished in all four limbs. Plantar response was flexor on both sides. Examination of sensory system, cerebellar function tests and fundus examination were normal. Patient was provisionally diagnosed to have acute flaccid quadriparesis predominantly proximal muscle weakness with diffuse toxic goiter.

Patient was investigated with complete blood count with peripheral smear, renal function test, random blood sugar, electrolytes (Na+, K+, Mg2+, Ca2+,Cl-,HCO3-), Creatine kinase -total, complete urine analysis, Fasting lipid profile, Thyroid profile (TSH, Free T3, Free T4), Chest X-ray, electrocardiogram (ECG), Ultra sonogram of thyroid gland. Preliminary blood & urine investigations showed Serum potassium was 2.6 milliequivalents per litre (mEq/L) (normal range: 3.5 – 5.5 mEq/L), and elevated Free T3 & Free T4 levels with markedly decreased TSH level (Free T3 - 26.24 pg/ml, Free T4 - 6.08 ng/dl, TSH - 0.02 µIU/ml). ECG showed sinus rhythm with heart rate of 110/ min, prominent U wave. Ultra sonogram thyroid showed diffuse enlargement with altered echo texture and hyper vascularity. Based on clinical features along with above relevant investigations, patient was diagnosed to have "Thyrotoxic periodic paralysis (TPP)".

Patient was treated with intravenous potassium administration with careful monitoring of potassium levels in view of Hypokalemic paralysis. Patient was also started on non-selective beta adrenergic blocking agent (propranolol) along with anti- thyroid agent (carbimazole). Muscle power improved to normal within 48 hrs after which he was switched over to

oral potassium supplementation. His Serum potassium increased to 3.5 mEq /L. Potassium supplementation was discontinued after achieving normal levels. Patient was discharged with anti-thyroid drug and beta blocker. He was advised to avoid strenuous exercise, high carbohydrate diet and high salt intake. He is now in asymptomatic euthyroid state on regular follow-up for past 1 year.

3. DISCUSSION

3.1 Epidemiology

Thyrotoxicosis presenting as flaccid paralysis in Asian male is extremely rare occurrence. Despite a much higher incidence of thyrotoxicosis in women, Thyrotoxic periodic paralysis (TPP) predominantly affects males of Asian origin. In this race it occurs 70 times more frequently in males than females and usually occurs between the ages of 20 and 40 years reflecting the age of onset of thyrotoxic Graves' disease [5]. The age of onset of TPP is usually in the third to fifth decade of life. But there are case reports with TPP in a young boy of 14 years [6]. The male-to-female ratio of TPP is estimated to be 20 to 1[7]. Our patient typically falls within expected range of age group.

3.2 Clinical Features

In some patients, TPP may be the initial presentation of thyrotoxicosis. Proximal muscle weakness of lower limb is often the first symptom noted. TPP manifests classically in early morning after a period of sleep. It is characterized by sudden onset of episodic painless muscle weakness and significant hypokalemia, which can be life threatening if untreated. The duration of attacks range from 2-36 hours and can be shortened by K+ supplementation in appropriate situations [8]. This patient also had onset of weakness typically in early morning.

3.3 Pathogenesis

Thyrotoxic periodic paralysis (TPP) is one of the causes of hypokalemic paralysis. Hypokalemia is the consequence of a rapid and massive shift of potassium from the extracellular into the intracellular compartment, mainly into the muscles. It is thought that thyroid hormone increases sodium-potassium ATPase activity, which drives potassium into cells, creating muscle membrane hyperpolarization. Thus, whole-body potassium stores are not depleted, just temporarily driven intracellularly. Because thyroid hormone-responsive elements are located upstream of genes encoding subunits of the sodium-potassium ATPase, thyroid hormone can increase activity via transcriptional and post-transcriptional methods [9].

It is proposed that activities increasing the release of epinephrine or insulin can trigger periodic paralysis attacks because they increase potassium drive into cells [10]. Known triggers include eating carbohydrate-rich meals, vigorous exercise, menstruation, alcohol intake, exposure to cold, corticosteroid therapy, administration of insulin or epinephrine, infections, emotional stress, trauma and consuming supplements containing tiratricol [11]. Possible trigger in our case was vigorous exercise since he is a manual labourer. We have excluded other triggers by means of history.

Even in the absence of physical examination findings, it is important to consider TPP in the differential diagnosis for patients who present with marked weakness and unexplained hypokalemia. Diagnosis and treatment are important because severe hypokalemia can lead to serious cardiac arrhythmias like high-degree atrio-ventricular block, ventricular tachycardia, and ventricular fibrillation, which are potential causes of mortality [12].

3.4 Differential Diagnosis

TPP is distinguished from other forms of periodic paralysis (especially hypokalemic periodic paralysis) with thyroid function tests. These are normal in the other forms, and in thyrotoxicosis the levels of thyroxine and tri-iodothyronine are elevated, with suppression of TSH. Our patient had markedly suppressed TSH with elevated free T3 and free T4 with profound hypokalemia which made the diagnosis certain.

The differential diagnosis should include Guillain-Barre syndrome (GBS), which also presents with lower extremity weakness and areflexia. In GBS, flaccid weakness is associated with normal serum potassium and thyroid profile with nerve conduction abnormalities. However, unlike Guillain-Barre syndrome, which has an estimated incidence of respiratory paralysis of 30%, TPP very rarely causes respiratory failure [13]. Bulbar, respiratory, and ocular muscles are usually spared in TPP. Our patient did not develop respiratory muscle weakness.

3.5 Management

Thyrotoxic periodic paralysis is a curable disorder that resolves when euthyroid status is achieved with anti-thyroid therapy. Hypokalemia in TPP is due to intra cellular shift of potassium, and not due to deficiency of potassium. Hence, correction of hypokalemia should be done with extreme caution with close monitoring of serum potassium levels because of the risk of hyperkalemia. Potassium supplementation must be discontinued once normal serum potassium level is achieved. The use of early non-selective beta-blocker therapy has been studied and shown to improve refractory hypokalemia by blocking the stimulation of the sodium-potassium ATPase activity [14]. Acetazolamide, which has been reported to decrease the frequency of paralytic attacks in familial hypokalemic periodic paralysis (FHPP), should never be given to patients with TPP as it may actually worsen the attack [15].

4. CONCLUSION

Thyrotoxic periodic paralysis is a potentially lethal manifestation of uncontrolled hyperthyroidism encountered commonly in Asian men in the age group of 20 to 40 years. It may be a first manifestation of thyrotoxicosis. It is often differentiated from similar clinical conditions with relevant history and thyroid profile. Early administration of beta blocker therapy and potassium supplementation helps to ameliorate hypokalemic paralysis and life threatening arrhythmias. Hypokalemia correction should be done with close monitoring of serum K+ levels to prevent rebound hyperkalemia. Achieving euthyroid state with anti-thyroid measures will prevent further episodes of recurrent hypokalemic paralysis.

CONSENT

No written consent was obtained from the patient since any visual content or ID information that could reveal the identity or violate the privacy of the patient was used in the article

ETHICAL CONSIDERATION

Institute ethical committee clearance has been obtained for this case report before publication.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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